

REMARKS

The following remarks are supplemental to those of the April 25 amendment and in response to the Advisory Action dated May 5, 2007 (“the Advisory Action”). In the Advisory Action, the Examiner contends that amendments submitted in the April 25 amendment may encompass new matter. In particular, the Examiner contends that Applicants point to support in the specification “(e.g., on page 21, lines 4-8)” but that no such support is found in the specification as directed. Applicants presume the Examiner’s objection to be to the indicated support in the specification for the amendments to claims 1, 23, 42 and 81-90, which is indicated in the April 25 amendment to be, e.g., page 21, lines 4-8; page 34, lines 15-16; page 60, lines 6-15; and page 135, lines 12-21 (see, the April 15 amendment at page 17, lines 16-17). Applicants respectfully disagree with the Examiner’s position.

In particular, claims 1, 23, 42 and 81-89 have been amended, in part, to recite, “An isolated IgG antibody...” Support for this amendment may be found at page 21, lines 7-8, which states, “[i]mmunoglobulin molecules can be of any type (e.g., IgG....”

Claims 1, 23, 42 and 81 were further amended, in part, to recite “... wherein said variable domain specifically binds FcγRIIB that is endogenously expressed on the surface of a human cell.” Support for this amendment may be found at page 60, lines 6-10, “[t]arget cells used in the ADCC assay of the invention include, ... Daudi cells ...” and lines 14-15, “[t]arget cells must be recognized by the antigen binding site of the antibody to be assayed,” and, in a specific example, at page 135, lines 12-13, “[a] double staining FACS assay was used to characterize the antibody produced from clones 2B6 and 3H7 in human B lymphocytes.” Further support may also be found, e.g., at page 56, lines 19-20, “[t]he invention relates to characterizing the anti-FcγRIIB antibodies of the invention for FcγRII-mediated signaling in human monocytes/macrophages;” at page 56, lines 30-32, “[t]he invention further encompasses characterizing the anti-FcγRIIB antibodies of the invention for their inhibition of FcγR-mediated phagocytosis in human monocytes/macrophages;” and at page 58, lines 4-5, “[i]n a preferred embodiment, the antibodies of the invention modulate FcγRIIB-dependent activity in human monocytes/macrophages.”

The above passages make clear that the invention describes the binding of the claimed antibody to its antigen, *i.e.*, FcγRIIB, that is endogenously expressed on the surface of a

human cell. Accordingly, the amendments to claims 1, 23, 42 and 81-90 do not constitute new matter.

CONCLUSION

Applicants respectfully request that the remarks made herein be entered and made of record in the instant application. If any issues remain in connection herewith, the Examiner is respectfully invited to telephone the undersigned to discuss the same.

Respectfully submitted,

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